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=> s (GABA(w)A or gamma(w)amino(w)butyric(w)acid(w)A)
L1       45722 (GABA(W) A OR GAMMA(W) AMINO(W) BUTYRIC(W) ACID(W) A)

=> s l1 and delta(w)subunit
L2         717 L1 AND DELTA(W) SUBUNIT

=> s l2 and (express? or immunohistochem? or immunoblot?)
L3         503 L2 AND (EXPRESS? OR IMMUNOHISTOCHEM? OR IMMUNOBLOT?)

=> s l3 and (A4B2D or alpha4beta2delta)
L4          10 L3 AND (A4B2D OR ALPHA4BETA2DELTA)

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5           7 DUP REM L4 (3 DUPLICATES REMOVED)

=> dis ibib abs l5 1-7

L5   ANSWER 1 OF 7     MEDLINE on STN
ACCESSION NUMBER:   2009048178     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 18762200
TITLE:              Novel compounds selectively enhance delta
                    subunit containing GABA A
                    receptors and increase tonic currents in thalamus.
AUTHOR:             Wafford K A; van Niel M B; Ma Q P; Horridge E; Herd M B;
                    Peden D R; Belelli D; Lambert J J
CORPORATE SOURCE:   Department of Molecular and Cellular Neuroscience, Merck
                    Sharp & Dohme Research Laboratories, The Neuroscience
                    Research Centre, Harlow, United Kingdom..
                    waffordke@lilly.com
CONTRACT NUMBER:    C509923 (United Kingdom Biotechnology and Biological
                    Sciences Research Council)
SOURCE:             Neuropharmacology, (2009 Jan) Vol. 56, No. 1, pp. 182-9.
                    Electronic Publication: 2008-08-13.
                    Journal code: 0236217. ISSN: 0028-3908. L-ISSN: 0028-3908.
PUB. COUNTRY:       England: United Kingdom
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
                    (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
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ENTRY MONTH: 200904
ENTRY DATE: Entered STN: 6 Jan 2009
Last Updated on STN: 3 Apr 2009
Entered Medline: 2 Apr 2009

AB Inhibition in the brain is dominated by the neurotransmitter gamma-aminobutyric acid (GABA); operating through GABA(A) receptors. This form of neural inhibition was presumed to be mediated by synaptic receptors, however recent evidence has highlighted a previously unappreciated role for extrasynaptic GABA(A) receptors in controlling neuronal activity. Synaptic and extrasynaptic GABA(A) receptors exhibit distinct pharmacological and biophysical properties that differentially influence brain physiology and behavior. Here we used a fluorescence-based assay and cell lines expressing recombinant GABA(A) receptors to identify a novel series of benzamide compounds that selectively enhance, or activate alpha4beta3delta GABA(A) receptors (cf. alpha4beta3gamma2 and alpha1beta3gamma2). Utilising electrophysiological methods, we illustrate that one of these compounds, 4-chloro-N-[6,8-dibromo-2-(2-thienyl)imidazo[1,2-a]pyridine-3-yl benzamide (DS1) potently (low nM) enhances GABA-evoked currents mediated by alpha4beta3delta receptors. At similar concentrations DS1 directly activates this receptor and is the most potent known agonist of alpha4beta3delta receptors. 4-chloro-N-[2-(2-thienyl)imidazo[1,2-a]pyridine-3-yl benzamide (DS2) selectively potentiated GABA responses mediated by alpha4beta3delta receptors, but was not an agonist. Recent studies have revealed a tonic form of inhibition in thalamus mediated by the alpha4beta2delta extrasynaptic GABA(A) receptors that may contribute to the regulation of thalamocortical rhythmic activity associated with sleep, wakefulness, vigilance and seizure disorders. In mouse thalamic relay cells DS2 enhanced the tonic current mediated by alpha4beta2delta receptors with no effect on their synaptic GABA(A) receptors. Similarly, in mouse cerebellar granule cells DS2 potentiated the tonic current mediated by alpha6betadelta receptors. DS2 is the first selective positive allosteric modulator of delta-GABA(A) receptors and such compounds potentially offer novel therapeutic opportunities as analgesics and in the treatment of sleep disorders. Furthermore, these drugs may be valuable in elucidating the physiological and pathophysiological roles played by these extrasynaptic GABA(A) receptors.

L5 ANSWER 2 OF 7 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2008317535 MEDLINE
DOCUMENT NUMBER: PubMed ID: 18480294
TITLE: Ketamine, but not phencyclidine, selectively modulates cerebellar GABA(A) receptors containing alpha6 and delta subunits.
AUTHOR: Hevers Wulf; Hadley Stephen H; Luddens Hartmut; Amin Jahanshah
CORPORATE SOURCE: Carl-Ludwig Department of Physiology, University of Leipzig, D-04103 Leipzig, Germany.
SOURCE: The Journal of neuroscience : the official journal of the Society for Neuroscience, (2008 May 14) Vol. 28, No. 20, pp. 5383-93.
Journal code: 8102140. E-ISSN: 1529-2401. L-ISSN: 0270-6474.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200806

ENTRY DATE: Entered STN: 16 May 2008
Last Updated on STN: 20 Jun 2008
Entered Medline: 19 Jun 2008

AB Phencyclidine (PCP) and ketamine are dissociative anesthetics capable of inducing analgesia, psychomimetic behavior, and a catatonic state of unconsciousness. Despite broad similarities, there are notable differences between the clinical actions of ketamine and PCP. Ketamine has a lower incidence of adverse effects and generally produces greater CNS depression than PCP. Both noncompetitively inhibit NMDA receptors, yet there is little evidence that these drugs affect GABA(A) receptors, the primary target of most anesthetics. alpha6beta2/3delta receptors are subtypes of the GABA(A) receptor family and are abundantly expressed in granular neurons within the adult cerebellum. Here, using an oocyte expression system, we show that at anesthetically relevant concentrations, ketamine, but not PCP, modulates alpha6beta2delta and alpha6beta3delta receptors. Additionally, at higher concentrations, ketamine directly activates these GABA(A) receptors. Comparatively, dizocilpine (MK-801 [(+)-5-methyl-10,11-dihydro-5H-dibenzo [a,d] cyclohepten-5,10-imine maleate]), a potent noncompetitive antagonist of NMDA receptors that is structurally unrelated to PCP, did not produce any effect on alpha6beta2delta receptors. Of the recombinant GABA (A) receptor subtypes examined (alphalbeta2, alphalbeta2gamma2, alphalbeta2delta, alpha4beta2gamma2, alpha4beta2delta, alpha6beta2gamma2, alpha6beta2delta, and alpha6beta3delta), the actions of ketamine were unique to alpha6beta2delta and alpha6beta3delta receptors. In dissociated granule neurons and cerebellar slice recordings, ketamine potentiated the GABAergic conductance arising from alpha6-containing GABA(A) receptors, whereas PCP showed no effect. Furthermore, ketamine potentiation was absent in cerebellar granule neurons from transgenic functionally null alpha6(-/-) and delta(-/-)mice. These findings suggest that the higher CNS depressant level achieved by ketamine may be the result of its selective actions on alpha6beta2/3delta receptors.

L5 ANSWER 3 OF 7 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2007429292 MEDLINE
DOCUMENT NUMBER: PubMed ID: 17395622
TITLE: Rat alpha6beta2delta GABAA receptors exhibit two distinct and separable agonist affinities.
AUTHOR: Hadley Stephen H; Amin Jahanshah
CORPORATE SOURCE: Department of Molecular Pharmacology and Physiology, College of Medicine, University of South Florida, Tampa, FL 33612, USA.
SOURCE: The Journal of physiology, (2007 Jun 15) Vol. 581, No. Pt 3, pp. 1001-18. Electronic Publication: 2007-03-29. Journal code: 0266262. ISSN: 0022-3751. L-ISSN: 0022-3751. Report No.: NLM-PMC2170852.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: (COMPARATIVE STUDY) Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200708
ENTRY DATE: Entered STN: 27 Jul 2007
Last Updated on STN: 10 Aug 2007
Entered Medline: 9 Aug 2007

AB The onset of motor learning in rats coincides with exclusive expression of GABAA receptors containing alpha6 and delta subunits in the granule neurons of the cerebellum. This

development temporally correlates with the presence of a spontaneously active chloride current through alpha6-containing GABAA receptors, known as tonic inhibition. Here we report that the coexpression of alpha6, beta2, and delta subunits produced receptor-channels which possessed two distinct and separable states of agonist affinity, one exhibiting micromolar and the other nanomolar affinities for GABA. The high-affinity state was associated with a significant level of spontaneous channel activity. Increasing the level of expression or the ratio of beta2 to alpha6 and delta subunits increased the prevalence of the high-affinity state. Comparative studies of alpha6beta2delta, alpha1beta2delta, alpha6beta2gamma2, alpha1beta2gamma2 and alpha4beta2delta receptors under equivalent levels of expression demonstrated that the significant level of spontaneous channel activity is uniquely attributable to alpha6beta2delta receptors. The pharmacology of spontaneous channel activity arising from alpha6beta2delta receptor expression corresponded to that of tonic inhibition. For example, GABAA receptor antagonists, including furosemide, blocked the spontaneous current. Further, the neuroactive steroid 5alpha-THDOC and classical glycine receptor agonists beta-alanine and taurine directly activated alpha6beta2delta receptors with high potency. Specific mutation within the GABA-dependent activation domain (betaY157F) impaired both low- and high-affinity components of GABA agonist activity in alpha6betaY157Fdelta receptors, but did not attenuate the spontaneous current. In comparison, a mutation located between the second and third transmembrane segments of the delta subunit (deltaR287M) significantly diminished the nanomolar component and the spontaneous activity. The possibility that the high affinity state of the alpha6beta2delta receptor modulates the granule neuron activity as well as potential mechanisms affecting its expression are discussed.

L5 ANSWER 4 OF 7 MEDLINE on STN
ACCESSION NUMBER: 20071844474 MEDLINE
DOCUMENT NUMBER: PubMed ID: 17351635
TITLE: Reversal of neurosteroid effects at alpha4beta2delta GABAA receptors triggers anxiety at puberty.
AUTHOR: Shen Hui; Gong Qi Hua; Aoki Chiye; Yuan Maoli; Ruderman Yevgeniy; Dattilo Michael; Williams Keith; Smith Sheryl S
CORPORATE SOURCE: Department of Physiology and Pharmacology, SUNY Downstate Medical Center, 450 Clarkson Avenue, Brooklyn, New York 11203, USA.
CONTRACT NUMBER: P30 EY013079-079003 (United States NEI NIH HHS)
R01 EY008055-09 (United States NEI NIH HHS)
R01 EY013145-04 (United States NEI NIH HHS)
R01 NS041091-04 (United States NINDS NIH HHS)
SOURCE: Nature neuroscience, (2007 Apr) Vol. 10, No. 4, pp. 469-77. Electronic Publication: 2007-03-11.
Journal code: 9809671. ISSN: 1097-6256. L-ISSN: 1097-6256.
Report No.: NLM-NIHMS18000; NLM-PMC1858651.
PUB. COUNTRY: United States
DOCUMENT TYPE: (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200706
ENTRY DATE: Entered STN: 28 Mar 2007
Last Updated on STN: 27 Jun 2007
Entered Medline: 26 Jun 2007
AB Puberty is characterized by mood swings and anxiety, which are often

produced by stress. Here we show that THP (allopregnanolone), a steroid that is released as a result of stress, increases anxiety in pubertal female mice, in contrast to its anxiety-reducing effect in adults. Anxiety is regulated by GABAergic inhibition in limbic circuits. Although this inhibition is increased by THP administration before puberty and in adults, during puberty THP reduces the tonic inhibition of pyramidal cells in hippocampal region CA1, leading to increased excitability. This paradoxical effect of THP results from inhibition of alpha4betadelta GABAA receptors. These receptors are normally expressed at very low levels, but at puberty, their expression is increased in hippocampal area CA1, where they generate outward currents. THP also decreases the outward current at recombinant alpha4beta2delta receptors, and this effect depends on arginine 353 in the alpha4 subunit, a putative site for modulation by Cl-. Therefore, inhibition of alpha4beta2delta GABAA receptors by THP provides a mechanism for the generation of anxiety at puberty.

L5 ANSWER 5 OF 7 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2007374227 MEDLINE
DOCUMENT NUMBER: PubMed ID: 17521848
TITLE: GABAA receptors in the thalamus: alpha4 subunit expression and alcohol sensitivity.
AUTHOR: Jia Fan; Pignataro Leonardo; Harrison Neil L
CORPORATE SOURCE: Department of Anesthesiology, Weill Medical College, Cornell University, New York, NY 10021, USA.
SOURCE: Alcohol (Fayetteville, N.Y.), (2007 May) Vol. 41, No. 3, pp. 177-85. Electronic Publication: 2007-05-23. Ref: 81 Journal code: 8502311. ISSN: 0741-8329. L-ISSN: 0741-8329.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200708
ENTRY DATE: Entered STN: 27 Jun 2007 Last Updated on STN: 16 Aug 2007 Entered Medline: 15 Aug 2007

AB The inhibitory neurotransmitter gamma-aminobutyric acid (GABA) has long been implicated in the anxiolytic, amnesic, and sedative behavioral effects of alcohol. A large number of studies have investigated the interactions of alcohol with GABA receptors. Many investigators have reported effects of "high concentrations" (50-100 mM) of alcohol on GABA-mediated synaptic inhibition, but effects of the "low concentrations" (1-30 mM) of alcohol normally associated with mild intoxication have been elusive until recently. A novel form of "tonic inhibition" has been described in the central nervous system (CNS) that is generated by the persistent activation of extrasynaptic gamma-aminobutyric acid type A receptors (GABAA-Rs). These receptors are specific GABAA-R subtypes and distinct from the synaptic subtypes. Tonic inhibition regulates the excitability of individual neurons and the activity and rhythmicity of neural networks. Interestingly, several reports show that tonic inhibition is sensitive to low concentrations of alcohol. The thalamus is a structure that is critically important in the control of sleep and wakefulness. GABAergic inhibition in the thalamus plays a crucial role in the generation of sleep waves. Among the various GABAA-R subunits, the alpha1, alpha4, beta2, and delta subunits are heavily expressed in thalamic relay nuclei. Tonic inhibition has been demonstrated in thalamocortical relay neurons, where it is mediated by alpha4beta2delta GABAA-Rs. These extrasynaptic receptors are highly sensitive to gaboxadol, a novel hypnotic, but insensitive to benzodiazepines. Tonic inhibition is absent in thalamic relay neurons

from alpha4 knockout mice, as are the sedative and analgesic effects of gaboxadol. The sedative effects of alcohol can promote sleep. However, alcohol also disrupts the normal sleep pattern and reduces sleep quality. As a result, sleep disturbance caused by alcohol can play a role in the progression of alcoholism. As an important regulator of sleep cycles, inhibition in the thalamus may therefore be involved in both the sedative effects of alcohol and the development of alcoholism. Investigating the effects of alcohol on both synaptic and extrasynaptic GABAA-Rs in the thalamus should help us to understand the mechanisms underlying the interaction between alcohol and sleep.

L5 ANSWER 6 OF 7 MEDLINE on STN
ACCESSION NUMBER: 2006067963 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16452673
TITLE: Delta subunit susceptibility variants E177A and R220H associated with complex epilepsy alter channel gating and surface expression of alpha4beta2delta GABAA receptors.
AUTHOR: Feng Hua-Jun; Kang Jing-Qiong; Song Luyan; Dibbens Leanne; Mulley John; Macdonald Robert L
CORPORATE SOURCE: Department of Neurology, Vanderbilt University Medical Center, Nashville, Tennessee 37212, USA.
CONTRACT NUMBER: NS33300 (United States NINDS NIH HHS)
SOURCE: The Journal of neuroscience : the official journal of the Society for Neuroscience, (2006 Feb 1) Vol. 26, No. 5, pp. 1499-506.
Journal code: 8102140. E-ISSN: 1529-2401. L-ISSN: 0270-6474.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200604
ENTRY DATE: Entered STN: 3 Feb 2006
Last Updated on STN: 28 Apr 2006
Entered Medline: 27 Apr 2006

AB Most human idiopathic generalized epilepsies (IGEs) are polygenic, but virtually nothing is known of the molecular basis for any of the complex epilepsies. Recently, two GABAA receptor delta subunit variants (E177A, R220H) were proposed as susceptibility alleles for generalized epilepsy with febrile seizures plus and juvenile myoclonic epilepsy. In human embryonic kidney 293T cells, recombinant halpha1beta2delta(E177A) and halpha1beta2delta(R220H) receptor currents were reduced, but the basis for the current reduction was not determined. We examined the mechanistic basis for the current reduction produced by these variants using the halpha4beta2delta receptor, an isoform more physiologically relevant and linked to epileptogenesis, by characterizing the effects of these variants on receptor cell surface expression and single-channel gating properties. Expression of variant alpha4beta2delta(R220H) receptors resulted in a decrease in surface receptor proteins, and a smaller, but significant, reduction was observed for variant alpha4beta2delta(E177A) receptors. For both variants, no significant alterations of surface expression were observed for mixed population of wild-type and variant receptors. The mean open durations of alpha4beta2delta(E177A) and alpha4beta2delta(R220H) receptor single-channel currents were both significantly decreased compared to wild-type receptors. These data suggest that both delta(E177A) and delta(R220H) variants may result in disinhibition in IGEs by similar cellular and molecular mechanisms, and in

heterozygously affected individuals, a reduction in channel open duration of delta subunit-containing GABAA receptors may be the major contributor to the epilepsy phenotypes.

L5 ANSWER 7 OF 7 MEDLINE on STN
ACCESSION NUMBER: 2006571671 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16844844
TITLE: Effects of ethanol on tonic GABA currents in cerebellar granule cells and mammalian cells recombinantly expressing GABA(A) receptors.
AUTHOR: Yamashita Megumi; Marszalec William; Yeh Jay Z; Narahashi Toshio
CORPORATE SOURCE: Department of Molecular Pharmacology and Biological Chemistry, Northwestern University Medical School, 303 E. Chicago Ave., Chicago, IL 60611-3008, USA.
CONTRACT NUMBER: R01 AA07836 (United States NIAAA NIH HHS)
SOURCE: The Journal of pharmacology and experimental therapeutics, (2006 Oct) Vol. 319, No. 1, pp. 431-8. Electronic Publication: 2006-07-14.
Journal code: 0376362. ISSN: 0022-3565. L-ISSN: 0022-3565.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200611
ENTRY DATE: Entered STN: 27 Sep 2006
Last Updated on STN: 3 Nov 2006
Entered Medline: 2 Nov 2006

AB The effects of ethanol on the GABA(A) receptors, which are regarded as one of the most important target sites of ethanol, are very controversial, ranging from potentiation to no effect. The delta subunit-containing GABA(A) receptors expressed in Xenopus oocytes were recently reported to be potently augmented by ethanol. We performed patch-clamp experiments using the cerebellar granule cells and mammalian cells expressing recombinant GABA(A) receptors. In granule cells, the sensitivity to GABA increased from 7 to 11 days in vitro. Furosemide, an antagonist of alpha6-containing GABA(A) receptors, inhibited GABA-induced currents more potently at 11 to 14 days than at 7 days. Ethanol at 30 mM had either no effect or an inhibitory effect on currents induced by low concentrations of GABA in granule cells. On alpha4beta2delta, alpha6beta2delta, or alpha6beta3deltaGABA(A) receptors expressed in Chinese hamster ovary cells, ethanol at 10, 30, and 100 mM had either no effect or an inhibitory effect on GABA currents. Ethanol inhibition of GABA(A) receptor was observed in all of the subunit combinations examined. In contrast, the perforated patch-clamp method to record the GABA currents revealed ethanol effects on the alpha6beta2delta subunits ranging from slight potentiation to slight inhibition. Ethanol seems to exert a dual action on the GABA(A) receptors and the potentiating action may depend on intracellular milieu. Thus, the differences between the GABA (A) receptors expressed in mammalian host cells and those in Xenopus oocytes in the response to ethanol might be due to changes in intracellular components under patch-clamp conditions.